

A Survey of the Potential Effects of Increasing UV-B Radiation on the Biosphere

30 September 1998

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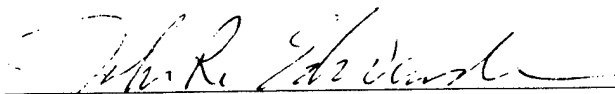
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This report has been reviewed by the Public Affairs Office (PAS) and is releasable to the National Technical Information Service (NTIS). At NTIS, it will be available to the general public, including foreign nationals.

This technical report has been reviewed and is approved for publication. Publication of this report does not constitute Air Force approval of the report's findings or conclusions. It is published only for the exchange and stimulation of ideas.



John R. Edwards, SMC/AXFV
Chief, Environmental Management Branch

To Readers of this Report:

This report is a revised re-issue of Aerospace Report No. TR-98(1306)-1, "A Survey of the Potential Effects of Increasing UV-B Radiation on the Biosphere," issued on 15 December 1997. That report was recalled for further review and comments within The Aerospace Corporation.

M. A. Kwok

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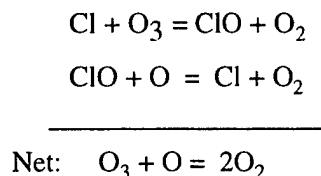
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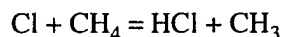
1. Background

Concern about the effect of chlorofluoromethanes on the stratospheric ozone layer began with the initial papers by *Rowland and Molina* (1974, 1975). The purpose of this document is to survey and evaluate the present state of knowledge about the possible environmental effects of changes in the ozone layer on humans, animals, and plants. This information may be useful in the future for environmental impact analyses due to human activity.

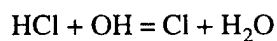
It is by now well established that the basic problem with introducing chlorine (or bromine) atoms into the stratosphere is that the halogen atom undergoes a sequence of fast chemical reactions that result in recombination of ozone and atomic oxygen and regeneration of the halogen atom:



Several other reaction cycles of this type have been shown to be involved. For more details, see *McElroy and Salawitch* (1989). The halogen atoms may be lost by reactions such as:



But they may also be returned to the cycle by reactions such as:



Thus, a chlorine atom will destroy the order of 10^5 ozone molecules before it ultimately diffuses back into the troposphere as HCl and rains out (*Rowland and Molina*, 1975). These reactions, by speeding up the loss of ozone, ultimately reduce ozone's steady-state concentration.

It was recognized very early that the principal problem caused by a reduction in the amount of ozone in the stratosphere would be an increase in ultraviolet (UV) light at the Earth's surface. This effect was difficult to document initially (see next section), but evidence for an increase in UV light is now very clear, and large increases are seen in the Antarctic as a result of the "ozone hole" formed each Austral spring.

Since UV light is readily absorbed by living tissue, and since light of this wavelength has a quantum energy strong enough to break chemical bonds, there was concern about its effects on living things.

This report is a review of the literature on the possible effects of increased UV light on plants, animals, and humans. At this time, it appears to be difficult to assign economic costs to the effects, but in some cases, risk factors may be assigned to some of the consequences.

2. Solar Radiation and "Amplification Factors"

By convention, solar ultraviolet radiation is divided into three wavelength bands: UV-A, 400–320 nm; UV-B, 320–290 nm, and UV-C, 290–190 nm. None of these wavelengths is visible to the human eye. The bands are, in fact, defined by the ozone layer itself. UV-A is not appreciably absorbed by ozone and reaches the Earth's surface attenuated only by light scattering. UV-B is just at the edge of the ozone layer's absorption and is, therefore, very sensitive to changes in the ozone column. UV-C is entirely absorbed by ozone and by oxygen, and does not reach the Earth's surface at all. It is found only in artificial light sources such as sterilizing lamps and electric welding arcs. For purposes of this report, the UV-A and UV-C radiation bands are of no concern because the amount of radiation in these bands will not change in response to changes in the amount of ozone.

Quantifying the amount of UV-B that reaches the Earth's surface is difficult, because there are so many complicating factors. The amount of radiation depends, of course, on the time of day, the season, and the cloudiness. Furthermore, the ozone column is comparatively less near the equator, and more at the poles, as shown in Figure 1. Recently, of course, "holes" in the ozone layer have appeared near the south pole, and some thinning has also been seen in the North as well. Figure 2 gives an indication of the natural variations in the ozone column as a function of time. The data in Figure 2 are taken from *Gleason, et al.*, and are one-week, area-weighted averages for 65°S to 65°N.

Figure 3 shows some of the time variation on a daily scale (from *CIAP, 1975*). In addition to these complications, the amount of radiation falls very rapidly at shorter wavelengths, which is a combined effect of the falling black-body radiation of the sun and the absorption by the ozone itself. The amount of UV energy is, in fact, small compared to the total visible and IR radiation, but because of its short wavelength, this radiation is very chemically active. If in fact, we consider the *total energy* in the UV-B band (320–290 nm), this is not very sensitive to small changes in the amount of ozone in the stratosphere. The problem is that living things are increasingly sensitive to the shortest wavelengths. Thus, a meaningful measure of the potential damage from UV-B must include a spectral weighting function to reflect the physiological impact of the radiation. The problem is illustrated in

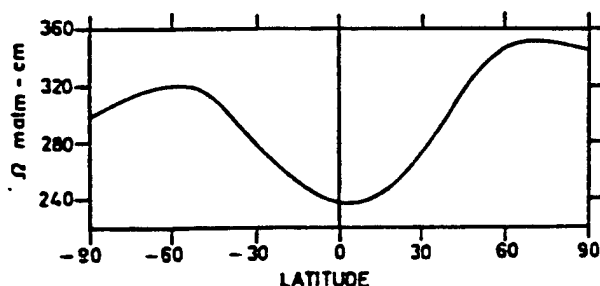


Figure 1. Total ozone column global average as a function of latitude. From *CIAP (1975)*.

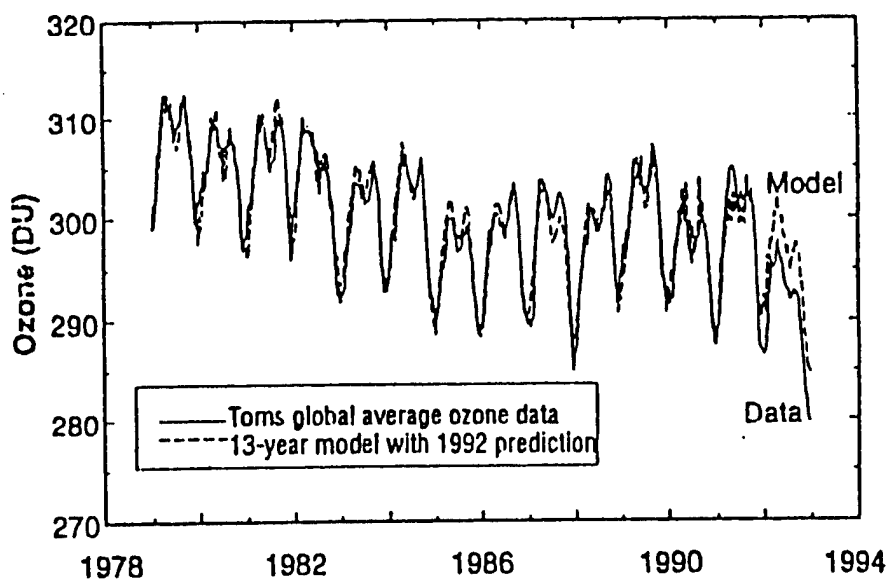


Figure 2. The annual average ozone amount for the latitude range 65°S to 65°N for 1 January 1979 to 31 December 1992 shown as a continuous time series (solid line). Each data point is a 1-week average. The annual, solar, and QBO cycles are clearly evident. In addition to the measured ozone time series, a statistical model is shown (dashed line) fitted to the 1979 to 1991 time period and extrapolated to 1992. From Gleason, et al., (1993).

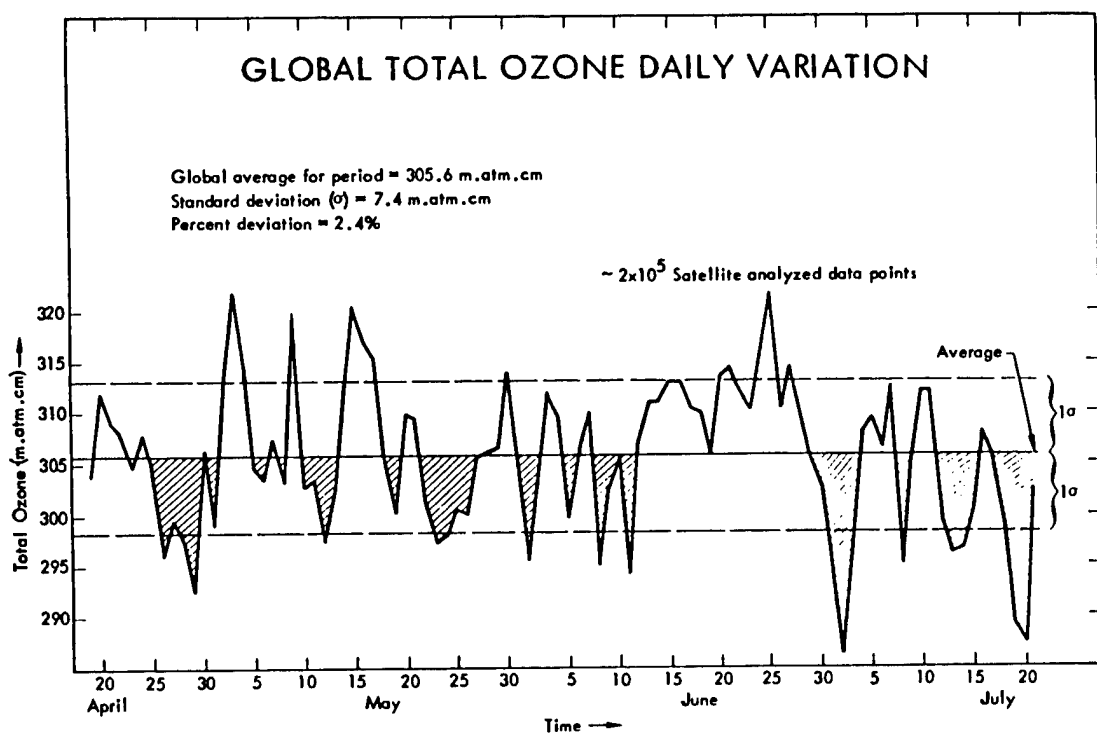


Figure 3. The daily variations of Global Total Ozone, as compiled from $\sim 2 \times 10^5$ satellite data points. From CIAP (1975).

Figure 4, which shows both the spectrum of light reaching the Earth's surface, and a typical action spectrum. The net effect of the radiation will be the overlap between the two spectra, shown in Figure 5. It can be seen that the overlap spectrum will be very sensitive to changes in the amount of ozone because both the radiation and the absorption are rapidly changing functions of wavelength.

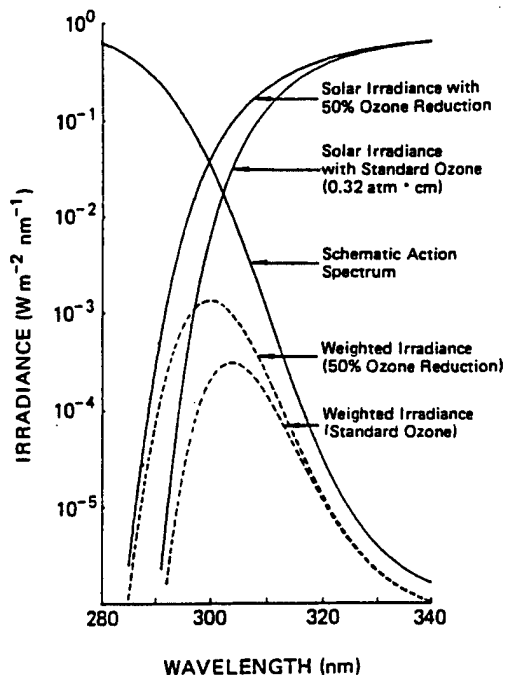


Figure 4. Semilog plot of a typical solar spectrum and a typical action spectrum, with the weighted overlap. Shown of normal and a 50% ozone reduction. From NAS (1979).

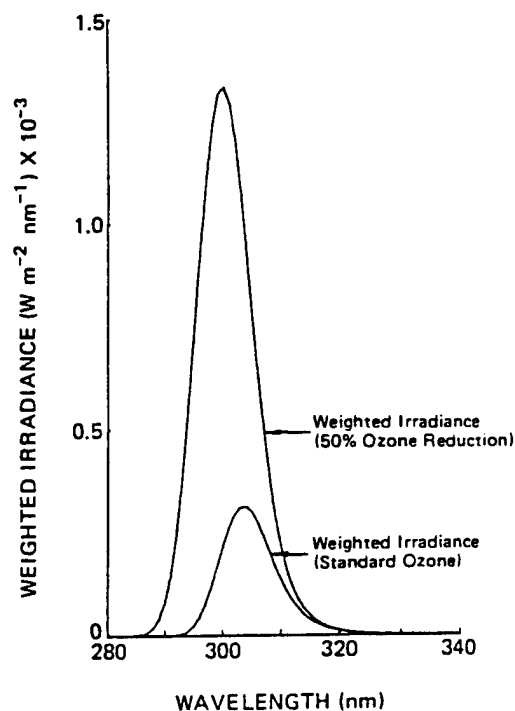


Figure 5. Weighted overlap spectra from Figure 2, plotted on a linear scale. From NAS (1979).

The irradiance overlap is dealt with by the idea of “radiation amplification factors” (RAFs). These appear in the literature in a number of forms, but the best defined appears to be the “two-part” RAF (see *Smith and Baker*, 1980 and *NRC*, 1979) given by:

$$\text{RAF} = (\text{BAF}) (\text{PAF}),$$

i.e., $\text{RAF} = (\text{biological amplification factor}) (\text{physical amplification factor})$

The $\text{BAF} = \% \text{Eff}/\% \text{DUV}$; or the percent change in a biological effect such as incidence rate of cancer, per percent change in DUV, or “damaging ultraviolet.”

The $\text{PAF} = \% \text{DUV}/\% \text{O}_3$; or the percent change in DUV per percent change in the average thickness of the ozone layer (a minus sign is understood). Thus,

$$\text{RAF} = \% \text{Eff}/\% \text{O}_3$$

These relationships are valid only for small changes, the order of 10%. Larger changes require the use of a logarithmic representation (*WMO*, 1994).

It will be appreciated that a great deal of averaging must go into the PAF for a global value to be meaningful since it depends on latitude, climate, season, time-of-day, and existing ozone levels. The World Meteorological Organization (1991) quotes a range of $\pm 16\%$ for an erythematous PAF (which they call RAF). Also note that “DUV” is not defined, but must be determined by the physiological weighting function chosen for the biological problem of interest. At first, it may seem unfortunate that biology must be factored into the PAF in this way. Why not just include the change in the total UV-B for a change in ozone? The answer is that without a weighting function, this is not a useful definition. The percent change in the total UV-B for a percent change in ozone is a very small number, whereas the percent in *damaging* UV (DUV) may be 1–2 or more times the percent change in ozone. Thus, the biological definition, while more difficult to calculate, is much more meaningful. Figure 6 shows some typical weighting functions for the DUV. These are also called action spectra since they must be determined by absolute measurements of some physiological response as a function of wavelength. Three examples shown are for sunburn (erythema), inhibition of photosynthesis, and the DNA absorption spectrum. It can be seen that the sunburn and DNA spectra are much steeper than the inhibition of photosynthesis spectrum. For this reason, the DNA spectrum has a much larger PAF—typically about 2. The DNA spectrum is believed to represent the action spectrum for skin cancer, although this cannot be known for certain in humans (*NAS*, 1982). Recent animal experiments suggest a somewhat lower PAF of 1.2 to 1.4 for non-melanoma skin cancer (*UNEP*, 1994). Averaging values for several recent surveys gives $\text{PAF} = 2.0 \pm 0.7$ (see Table 1); the photosynthesis inhibition PAF is less than 1 because it includes longer wavelengths.

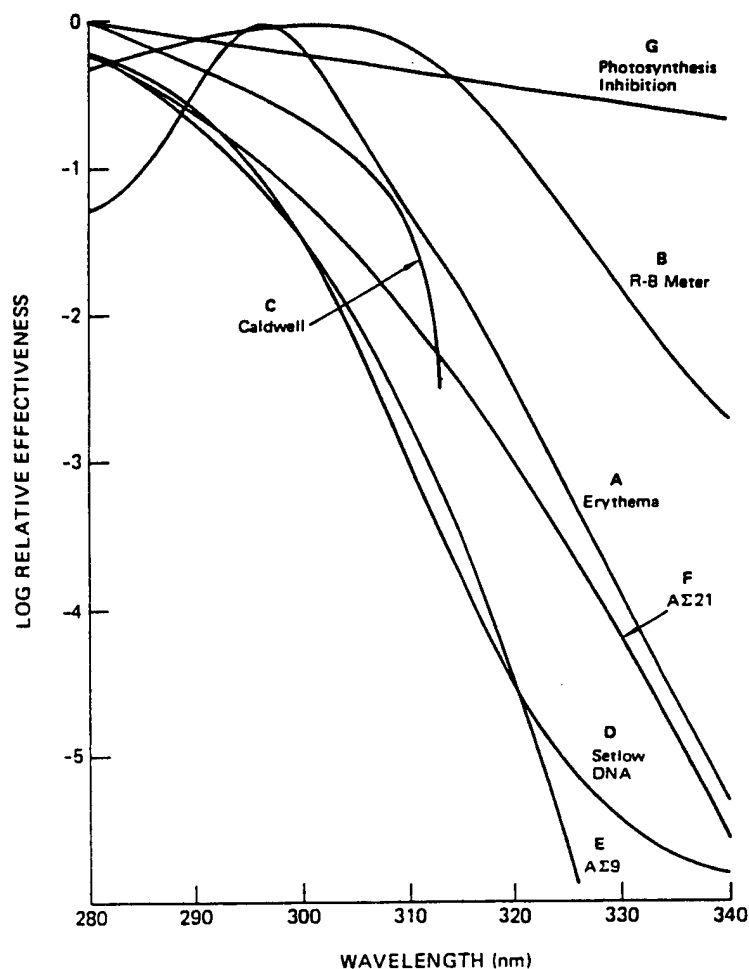


Figure 6. Some typical weighting spectra for biological damage. From NAS (1979).

Table 1. Summary of Amplification Factors

Factor	Value	Reference
PAF (DNA)	2.7	Gerstl, et al. (1981)
PAF (DNA)	2.0	NRC (1982)
PAF (DNA)	1.3	UNEP (1994)
BAF (NMSC)	2.4	NRC (1982)
BAF (NMSC)	2.0	NRC (1983)
BAF (NMSC)	2.0	Henrikson, et al. (1990)
BAF (NMSC) (*)	1.6	UNEP (1994)
Averages		
PAF	±0.7	
BAF	±0.3	
RAF = PAF x BAF	4.0±1.5	

* Weighted value: 80% BCC = 1.4; 20% SCC = 2.5±one standard deviation. NMSC = non-melanoma skin cancer; BCC = basal cell carcinoma, SCC = squamous cell carcinoma.

In this same area, we also show a curve marked "R-B" in Figure 6. This shows the instrumental sensitivity of the traditional Robertson-Berger (RB) UV meters. This curve is considerably broader than the DNA curve and, as a consequence, gives a smaller PAF. For example, the PAF at 40°N for the R-B curve is about 0.8, but for the DNA curve, it is about 2.3 (NRC, 1982). The instrument is, therefore, less sensitive to small changes in stratospheric ozone, and it may be for this reason that the earliest attempts to show an increase in ground-level DUV had negative results (Scotto, *et al.*, 1988; see WMO, 1991). More recent studies have documented a DUV increase (Kerr and McElroy, 1993; Kerr, 1994; Kerr *et al.*, 1994; Herman, *et al.*, 1996), especially in Antarctica (Frederick and Alberts, 1991). There have been suggestions that increases in tropospheric ozone will tend to offset the decrease in stratospheric ozone (Bruhl and Crutze, 1989; Varotsos, 1994), but since 90% of the ozone column is in the stratosphere (Madronich, 1992), this effect cannot go on indefinitely. Similarly, the ameliorating effects of aerosols are expected to level off in the future (Liu, *et al.*, 1991; Lubin and Jensen, 1995).

The BAF is an additional biological factor to reflect the fact that there may be *nonlinearities* in the physiological response to DUV. Thus, for example, if the biological studies find a power law of the sort: Effect \sim (DUV)ⁿ, then $BAF = \partial(\%Eff)/\partial(\%DUV) = n$.

This in fact appears to be the case for skin cancer, with "n" ranging from 2 to 4. (See below). The BAF may depend on factors such as skin pigmentation and the disease, but will not depend on latitude, season, time of day, or cloudiness.

3. Effects on Humans

We discuss effects on humans first, not only because of the potential importance, but also because these appear to be the best documented effects of UV radiation. In dealing with human effects, obvious ethical problems prevent the use of controlled experiments. This means that we must appeal to other types of reasoning than those typically found in papers about the physical sciences. Epidemiology, which includes study of the geographical and life-style patterns of disease incidence, is the main tool at our disposal. While this is not as precise as controlled laboratory experiments, it can still be quite persuasive.

Some of the recognized harmful effects of UV on humans include:

1. Non-melanoma skin cancer.
2. Melanoma skin cancer (may involve co-factors).
3. Immune inhibition.
4. Skin deterioration.
5. Cataracts.

There is also one well-known beneficial effect, which is the synthesis of Vitamin D₃ (the synthetic form is called D₂). This is presently adequately available in foods.

In this section, we will focus only on the first two harmful effects. Immune inhibition has been demonstrated in mice, but the quantitative aspects are not clear. Immune inhibition may play a role in skin cancers, but there is no latitude dependence for cancers other than skin cancer. In any case, the epidemiological treatment of skin cancer inherently includes all effects of sunlight. Skin deterioration due to sunlight is well documented, but since it is not fatal, we will not discuss it further. There appears to be a controversy about cataracts. Some sources state that cataracts are caused by UV-A radiation, and therefore, changes in ozone will not affect cataracts (*NRC*, 1982). A more recent paper (*Taylor and McCarty*, 1996) states that UV-B does cause cataracts. Since this is never a fatal condition (although it causes about 50% of all blindness) and since there is not much quantitative data, we will not treat it further.

Non-melanoma skin cancer (which includes basal- and squamous-cell carcinomas) is the most common form of all cancers, but has a low fatality rate. The epidemiological evidence for UV being a causative factor in non-melanoma skin cancers is as follows (*NAS*, 1976; *WMO*, 1994):

1. There is a striking increase with decreasing latitude. The incidence rate (race-corrected) is 4 times higher in Albuquerque than in Seattle, for example.

2. The cancers are most often found on areas of the body exposed to the sun.
3. The incidence is higher in people with outdoor occupations, and historically higher in men than in women.
4. The incidence increases with age.
5. Supported by animal studies.

In addition to these factors, there are the additional suggestive factors:

1. Incidence has risen 16-fold since the 1930's (see Figure 7.) This is likely due to the increase in sunbathing since that time, although other factors cannot be ruled out (NAS, 1976; Urbach, 1984; Long, 1996). Urbach points out that there is a pronounced cohort effect in which younger generations have a higher incidence at the same age as older generations (Urbach, 1984).
2. Incidence is inversely proportional to skin pigmentation. The incidence may vary from 10 to 70 times less than the maximum susceptibility as the skin pigmentation increases, and this is linearly proportional to the UV-B absorption of the skin (NAS, 1982). Furthermore, when the skin is protective, the cancer is usually found on the less pigmented parts of the body. Albino persons are especially susceptible. These effects appear to be a direct result of shielding of the DNA by melanin pigment.(NAS, 1983)

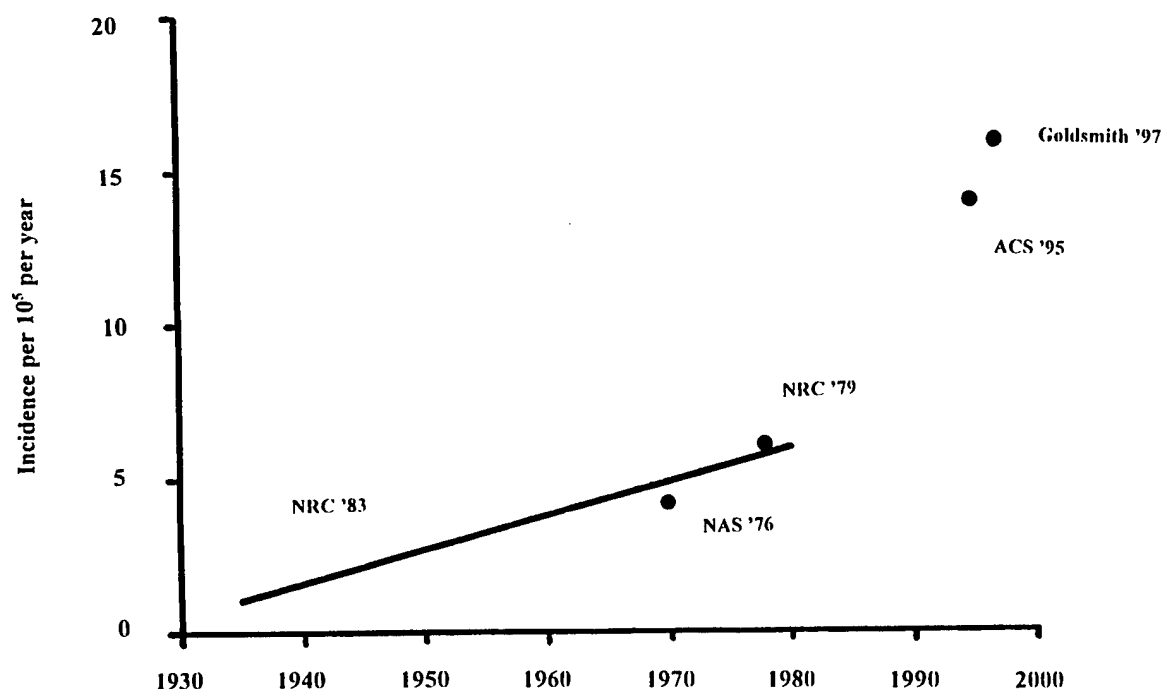


Figure 7. Incidence rate of malignant melanoma in the U.S. from 1935–1998. Data taken from NAS (1976), NRC(1979), NRC (1983), ACS (1995), Goldsmith (1997).

For melanoma, the same factors apply except for the occupational factor and there are no animal studies. The differences between different peoples is less (Urbach, 1984). The occupational factor is an apparent paradox: indoor workers have the highest incidence. There is a rationale for this, which we shall call the "vacation theory" (Urbach, 1984). The theory is that the indoor workers are exposed to sunlight all at once in midsummer, when they have no initial protective tan. The intense UV on unprotected skin is thought to lead to melanoma. Efforts to test the vacation theory have been inconclusive, but the theory is consistent with the idea that melanoma is a result of a short, intense exposure to sunlight, rather than long-term, chronic exposure. The evidence for this is that only 10% of melanoma cases show evidence of chronic sun damage, and the incidence may appear early in life. There may also be cofactors such as chemical exposure or genetic effects (Urbach, 1984). While these facts complicate the understanding of melanoma, they do not rule out the importance of UV-B.

With regard to the relationship between UV-B and melanoma, I will quote the 1994 UNEP report:

Cutaneous melanoma in humans may well have a multifactorial etiology. Although UV radiation is likely to play a dominant role (e.g., initiating precursor lesions during youth and suppressing immunity to the tumor cells as a result of a sunburn in the final stages of tumor development), other factors may affect the expression of the UV effect.

Figure 8 shows the results of a study in Norway (Henriksen, et al. 1988, 1990) that is as close as one can get to an experiment on humans. The researchers divided the country of Norway into four horizontal bands of latitude, which have appreciably different amounts of UV insolation. The statistics for skin cancer incidence (both kinds) were then plotted as a function of DUV, as shown in Figure 8. Since Norway has a pretty uniform population make-up, and presumably similar life-styles, the results are quite believable. The Figure shows a clear non-linearity, which corresponds to a BAF of 2.

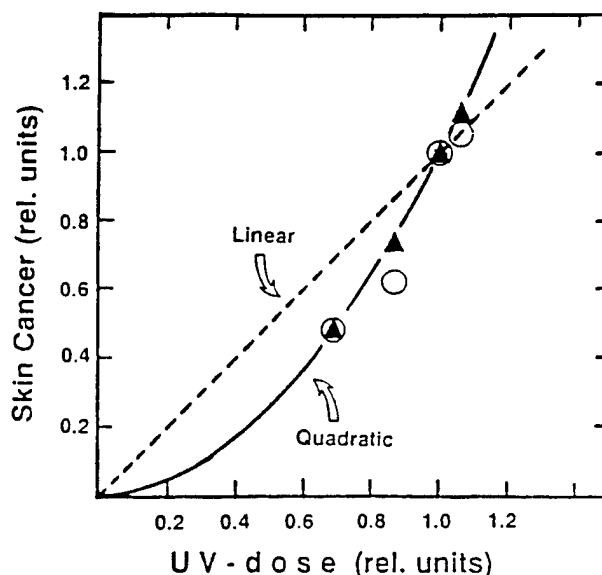


Figure 8. Skin cancer in Norway as a function of latitude, showing a clear quadratic relationship. Solid symbols are for non-melanoma and open symbols are for melanoma. From Henrikson, et al. (1988, 1990).

More latitude-dependent statistics are shown in Figure 9 (NRC, 1979). These are plotted as a function of latitude, but if we consult Figure 10 (ibid.), we see that the DUV is a roughly linear function of latitude for the range given (25°N to 45°N). Figure 11 shows a log-log re-plot of the incidence data as a function of DUV (for DNA spectra). The slopes of these lines gives the exponent in a power-law relationship for melanoma:

Thus, these data suggest a BAF of 3.5 ± 0.8 for melanoma, and higher numbers for non-melanoma. These numbers may be too high to the extent that "lifestyle" effects, i.e., increased exposure to the sun, increase the cancer incidence at lower latitudes; or too low to the extent that there may be possible benefits from the increased non-UV solar radiation (light-assisted DNA repair) at lower latitudes (NRC, 1982). Although the correct average number is not clear at this time, we propose that reasonable numbers would be 2.0 ± 0.7 for the PAF and 2.0 ± 0.3 for the BAF, giving an overall RAF for skin cancers of 4.0 ± 1.5 . These numbers are summarized in Table 1.

The standard deviations in the PAF and BAF are based on the values given in Table 1. The standard deviation in the RAF is based on a standard root-mean-square propagation of errors treatment of the two components.

In 1995, the average incidence for non-melanoma skin cancers in the United States was about 300 per 10^5 people per year, and about 14 per 10^5 people per year for melanoma (Long, *et al.*, 1996). The incidence is rising at about 3% per year. The long-term fatality rate from melanoma is about 30%, and the long-term fatality rate for non-melanoma skin cancer is about 1% (NRC, 1982). Recent diagnosis estimates for the United States as a whole for the year 1995 are 800,000 cases of non-melanoma and 34,000 cases of melanoma, with 7,200 melanoma deaths and 2,100 non-melanoma deaths, for a total of 9,300 deaths (Long, *et al.* 1996). The annual number of fatalities is a smaller percentage of the annual incidence than the long-term fatality rate because the rate of incidence is increasing with time. Reported figures on the annual melanoma incidence rate have a standard deviation of about ± 600 in recent years (ACS website, 1998).

The world population is on average much less susceptible to skin cancer than the U. S. population. An estimate, correcting for the protective effects of melanin in the skin, of the world incidence of skin cancer deaths would be 5.5 times as many as the U.S. This is based on assuming the U.S. rate for the populations of Australia, Canada, Europe, Russia, and Ukraine; $1/10^{\text{th}}$ that rate for the populations of Asia and South America; and $1/70^{\text{th}}$ that rate for the populations of Africa. This gives a total of about 40,000 melanoma and 11,000 non-melanoma deaths. If we assume an RAF of 4.0 ± 1.5 for both kinds of skin cancer, this suggests that a 1% change in the ozone layer would cause a $4.0 \pm 1.5\%$ change in the number of deaths, or $1,600 \pm 600$ melanoma and 440 ± 170 non-melanoma deaths. At this time, it is believed that the effect of UV-B on melanoma is not proven, but the effect on non-melanoma is well established (UNEP, 1994).

Other estimates of the number of additional cancer deaths due to a possible decrease in the ozone layer appear in the 1976 NAS review and in the EPA document of 1987. The NAS estimate appears to be based on an RAF of 2, and the EPA estimate on a range of RAF of 4.8 to 7.6 for non-melanoma and an RAF of 1.0 to 2.0 for melanoma. The EPA fatality estimate is based on a 24% fatality rate for

melanoma, and a 2% fatality rate for non-melanoma, giving overall about a factor of 2 greater fatality rate than the estimate made in this report.

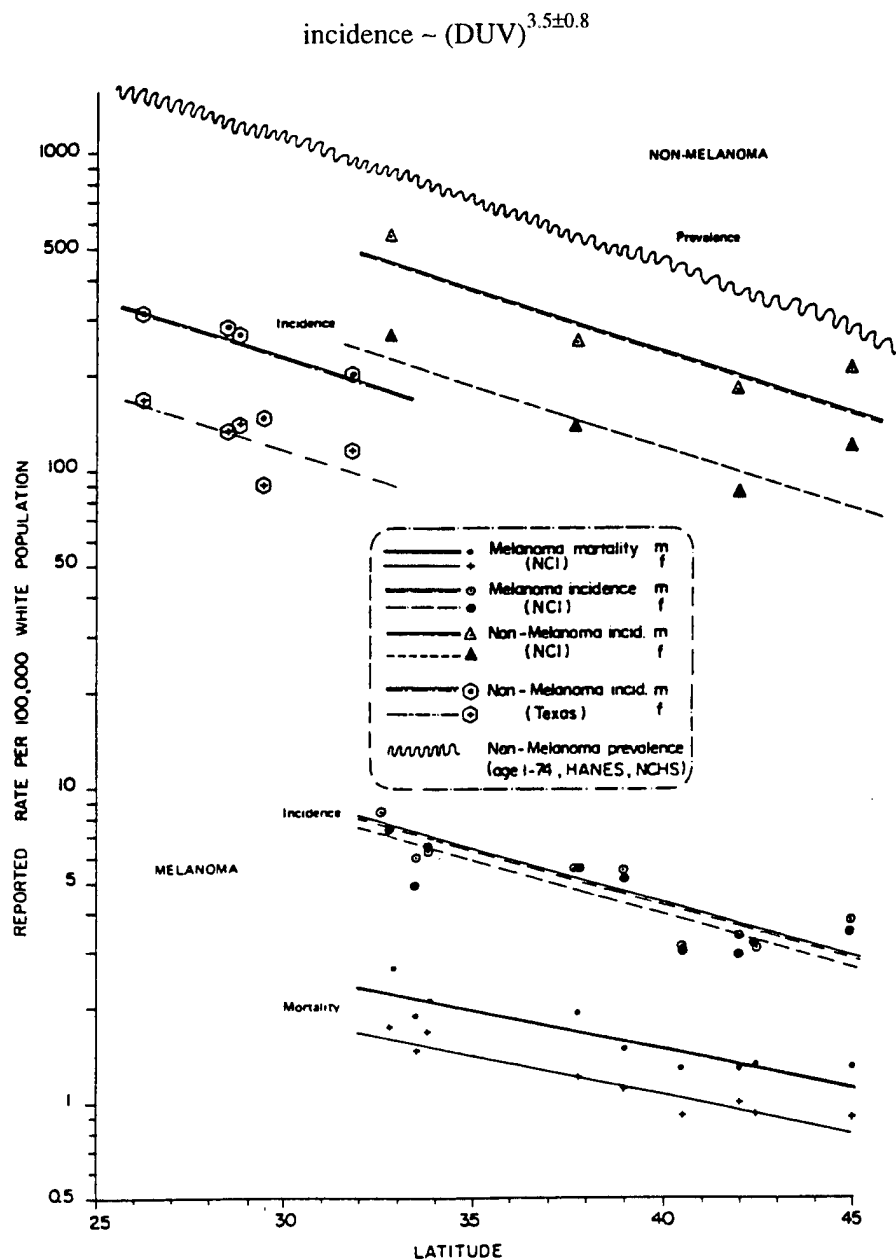


Figure 9. Reported skin cancer rates among whites as a function of latitude. (Sources: melanoma mortality from *Mason and McKay* (1974), melanoma incidence from National Cancer Institute (1974), nonmelanoma skin cancer incidence (NCI) from *Scotto, et al.* (1974), nonmelanoma skin cancer incidence (Texas) from *Mac Donald* (1974) and prevalence of nonmelanoma skin cancer based on preliminary data from the Health and Nutrition Examination Survey of the National Center for Health Statistics (*McDowell*, 1974). From NRC (1979). (Note: racial terms used in this and other references are those of the original authors.)

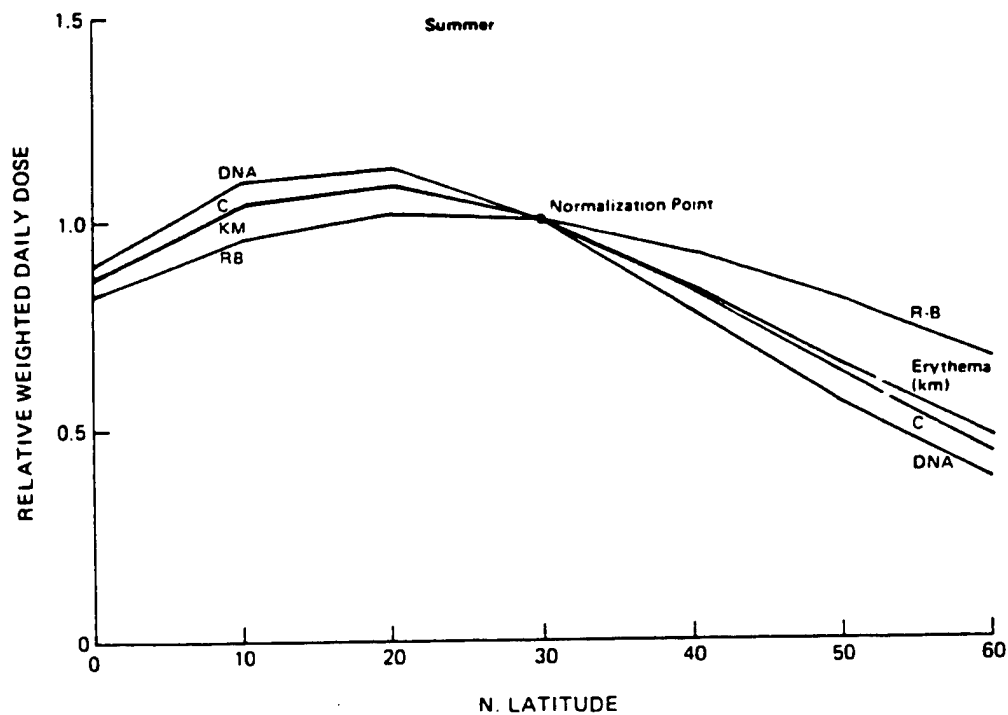


Figure 10. Relative daily doses at different northern latitudes at the summer solstice, calculated with different weighting functions. values have been normalized to the daily dose at 30°N latitude. From *NRC* (1979).

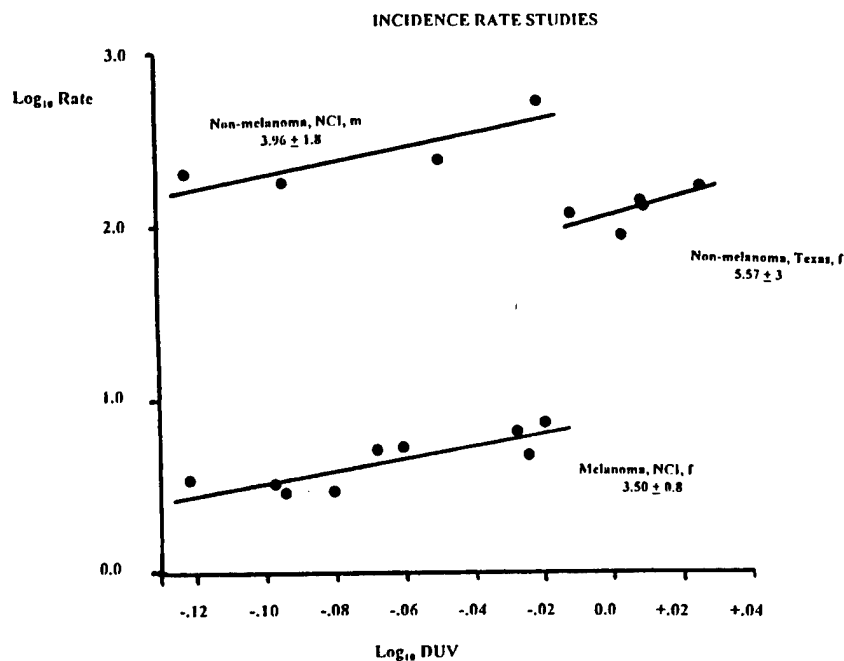


Figure 11. Log-log plot of data from Figure 9 as a function of DUV, taken from Figure 10. Three studies are shown. The slopes quoted will be equal to the exponential dependence of the incidence rate on the DUV.

4. Effects on Land Organisms

4.1 Cultivated Plants

Cultivated plants account for most of the world's food supply, and, therefore, even a small perturbation from increased UV-B could be of great economic significance. When concern grew about a possible increase in UV-B in the mid-1970's, a number of research studies were started on the effect of UV radiation on plants. In most of these studies, as in some earlier work, artificial radiation was used on plants in greenhouses. The early results were that roughly 20% of crop plants were sensitive to *existing* UV levels, 20% were insensitive to levels up to 4 times existing levels, and the remaining 60% were intermediate. More recent reviews suggest that 30% to 50% of all species are deleteriously affected by UV-B (*Teramura and Sullivan, 1994*). It has been shown that greenhouse studies tend to overestimate the sensitivity of plants because the visible light levels are reduced in a greenhouse. Visible light apparently has the ability to activate repair mechanisms in the plant, so that plants grown in the field are considerably less sensitive to ultraviolet. Furthermore, it proved difficult to duplicate the expected changes in solar radiation with artificial light sources. Subsequent work showed that great attention needed to be paid to getting a realistic spectrum, which happens to be difficult and expensive. An example of the early mistakes was the use of mercury lamps (254 nm). This wavelength is not found in ground-level sunlight at all! Some elaborate solutions to this problem have been tried, including using ozone light filters with sunlight (*Tevini, 1991*).

Plant studies are further complicated by a lack of reciprocity (*Cullen and Neale, 1994*), which means that studies must be done at close to the natural radiation intensities.

In spite of these problems, field studies have shown some sensitivity of plants to UV radiation. There is great variability in response not only between species, but also between varieties of the same species (cultivars). The responses also vary greatly, and may include yield; but also may involve more subtle changes such as leaf size, photosynthesis rate, resistance to diseases and insects, etc. It is interesting to note that some plants actually grow better in greenhouses because the plants are not optimized for existing UV levels in sunlight.

An example of the sensitivity of plants to UV light is the data shown in Figure 12, which is a plot of some tabulated data by *Tevini (1993)*. This plot shows two measures of cucumber growth—leaf area and stem length—as a function of “effective latitude,” which is a measure of UV content of the light. Cucumber is particularly sensitive to UV in both greenhouse and field studies. It can be seen that the growth is only about 50% to 60% as much at the equator as it is at 70°N.

Studies of this kind show that sugar beets, tomatoes, mustard, and cucumbers are sensitive to UV-B, whereas peanuts, peas, potatoes, and sorghum are not sensitive (*NRC, 1979*). With rice, corn, squash, and soybeans, the response depends on the variety (*Tevini, 1993*). Many of the reports are contradictory about the sensitivity of individual species to UV-B.

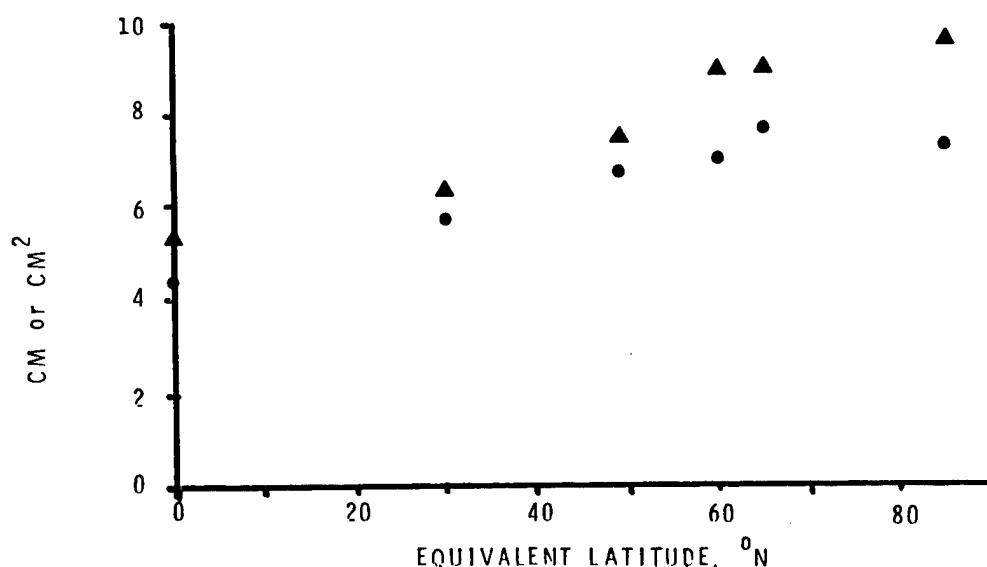


Figure 12. Effect of DUV changes on growth of cucumber seedlings, plotted as stem length and leaf area as a function of equivalent latitude. ● = stem length; ▲ = leaf area. Plotted from data tabulated by *Tevini* (1993).

Reviews of the plant studies suggest that any environmental response of plants to a change in UV is likely to be complex. For example, small changes in leaf size may alter the ability of weeds to grow around the plants of interest. Small changes in resistance to insects or disease or in the length of the growing season could cause large changes in yield. Studies on UV and plant diseases show that fungal and viral diseases may be inhibited or stimulated by UV, depending on the system. As a rule, however, the host plants appear to be harmed more by UV than the pathogens are (*Manning and Tiedemann*, 1995). In the Manning and Tiedemann paper, the use of Mylar UV filters in all greenhouses is suggested as a beneficial change. Because the different species vary widely in their response to UV—including a beneficial response in some case—the most likely thing to happen in the field is that there will be a change in the relative populations of the species (*Caldwell and Flint*, 1994). Some studies have been performed on the competitive balance between cultivated species and “weeds.” These studies have shown that sometimes the crop wins and sometimes the weeds win (*Runeckles and Krupa*, 1994). Studies have also been done on multiple stress factors such as heat, drought, or elevated local ozone (*Teramura*, 1990). The studies show that the stress factors are additive, but not synergistic. Teramura concluded that factors such as heat or drought could easily mask the effects of elevated UV-B. On the other hand, rapid fluctuations in the UV caused by tropospheric weather could be additionally stressful for plants (*Teramura and Sullivan*, 1994).

Because of all the complicating factors, and because managed agriculture may be able to compensate for changes in the environment, we conclude that it is not yet possible to assign a cost to potential crop loss from increased UV-B. Nevertheless, it should be kept in mind that the value of crops worldwide is extremely large, and even small perturbations could have large costs. Some estimates about economic damage appear in the book by Worrest (1986). World wheat, corn, and rice production are each roughly 500 billion tons a year, and U. S. exports of these products are over \$10 billion a year (*Johnson*, 1997).

4.2 Wild Plants

By "wild plants" we essentially mean forest trees, which account for 80% of the terrestrial productivity and which have great economic value. Trees are long-lived and therefore will have to face any UV stress for many seasons. Also, we have less control over them than we do with cultivated annual crops. Studies with trees are also more difficult because of their size and longevity, although a number of seedling studies have been carried out.

One potential problem with trees, which we will also see with the phytoplankton (see below), is that they may produce a connection between the ozone layer problem and the greenhouse effect. A reduction in productivity—the fixing of carbon dioxide by plants—could increase CO₂ levels. Thus, the ozone problem could indirectly contribute to increasing surface temperatures.

Teramura (1990, 1994) has reviewed the few studies on conifers. Of 15 species studied, 7 were harmed by UV, 5 were unharmed, and 3 improved. One of the most sensitive to harmful effects was the Loblolly pine, an economically important tree in the Southeast of the U.S. used in pulp production. This species showed a 40% mass loss under increased UV radiation (unspecified, but probably corresponding to a 25% ozone loss, the amount used in *Teramura's* previous studies). Since these were greenhouse studies, the effects may be exaggerated. Subsequent field studies on Loblolly showed that after three years, a 25% ozone loss equivalent caused a 17% to 19% loss of biomass in three out of four seed types. Similar results were seen for a 16% equivalent ozone reduction.

A few studies on other natural plants such as shrubs, has shown that transplanting from low-UV regions such as the Arctic to high-UV regions such as the equator, or going from low elevation to high elevation is generally deleterious (*Teramura*, 1990). In another experiment, seeds from 132 native and introduced plants were taken from various elevations in Hawaii and then grown under high UV (40% ozone reduction) in a greenhouse. Interestingly, only 8% of the species from 0 to 500 m elevation were able to tolerate the higher UV, but all species from over 2,000 m elevation were unaffected. Thus, by selection or adaptation, high-elevation species are more UV tolerant.

As in the case of crop plants, the consequences of a small ozone change on the world's natural plants may be subtle and complex. It is difficult to assign an economic cost at this time. A significant interaction with the greenhouse effect is possible.

It is interesting to consider the special problems encountered with the design of research programs in the area of global ecological impacts. It appears that a hierarchy of experiments is necessary in order to understand the effects of changes in large systems. To illustrate, consider an experiment to understand the effect of light on a plant. A first step would be to use pure monochromatic light and shine it on the plant. The results of such an experiment could be very well defined, but would have little relevance for the environment. Plants use a range of wavelengths of radiation to do many things, including repair of radiation damage. Thus, experiments with something closer to the solar spectrum would be more relevant, if not as sharply defined. Suppose further that an experiment gives an exact understanding of the effects of changes in solar radiation on the plant. This, it turns out, may have little bearing on the behavior of the plant in the environment. The UV radiation may alter the response of the plant to diseases or pests, or it may change the length of its growing season. The response in the environment may depend on the competition with other plants, i.e., on the differential effects of

radiation on its neighbors. Lastly, even a perfect understanding of the effects of UV radiation in the environment may be rendered useless if the radiation has other, global effects such as a change in cloud cover, precipitation patterns, temperatures, and so on. Thus, a hierarchy of experiments may be necessary to sort out what is happening.

4.3 Animals

The effects of increased UV radiation on wild and domesticated animals is not thought to be of great economic significance. The reason is twofold. First, most animals are protected from UV by fur or hair. Second, the few problems that are seen in cattle and sheep are eye and nose cancers that are infrequent or too late in life to have an economic impact (*NRC*, 1982).

5. Effects on Marine Organisms—Phytoplankton and Zooplankton

“Plankton” refers to those creatures too small to swim any great distance, thus they are those that drift with the ocean currents. Most are microscopic, although the zooplankton include the important larval stages of fish and shellfish. Adult fish are probably too deep to be at risk, but they depend on plankton for food. Phytoplankton are important because they are the base of a very short food chain:

Phytoplankton \Rightarrow Krill \Rightarrow all higher species

This chain is non-linear, with the fish production varying roughly as the phytoplankton production to the 1.5 power (*Haeder, 1994*). Since about 30% of all protein for human consumption is derived from the sea, our food supply is very dependent on the phytoplankton.

Furthermore, phytoplankton account for roughly half of the primary productivity (the rate of carbon dioxide fixation) in the world (104 billion tons of carbon per year vs 100 billion tons for all the land plants) (*Haeder, 1993*). Thus, a 10% decrease in productivity of these plants would be equivalent to all of the fossil fuel burning per year (10 billion tons).

When research began in this field, the researchers thought that it would simply be necessary to raise the UV intensity until deleterious effects were observed. To their astonishment, it was found that *existing levels* of UV are quite toxic. Indeed, it was soon discovered that since most previous experiments on phytoplankton were performed in glass containers, most such experiments had overestimated the productivity of such organisms in the open ocean (*Worrest, 1982*). In some cases, the productivity of phytoplankton is 2 to 4 times higher if natural UV-B and UV-A are excluded. (*Haeder, 1994*).

At this point, it should be mentioned that pure water is quite transparent to ultraviolet radiation. The main cause for attenuation is the presence of organics or light scattering material. Thus, the transmission is highly variable, but penetration of UV-B into the upper few meters of the ocean is significant. Figure 13 (*NRC, 1983*) shows some typical attenuation coefficients for seawater as a function of wavelength. A characteristic value for the attenuation of UV-B is about 1 m^{-1} . A review by *Haeder (1993)* points out that phytoplankton in fact flourish about 1.5 m below the surface of the ocean, with the population at that depth being over 5 times that at the surface. The phytoplankton appear to have the ability to descend to lower depths during periods of high light intensity to improve their survival. However, they respond only to visible light, and not UV, so an increase in UV is not something they could adapt to readily (*NRC, 1976*). *Haeder (1994)* also points out that marine productivity dips in mid-summer, which could be due to the higher irradiation, but there may be other causes for this, such as a lack of nutrients.

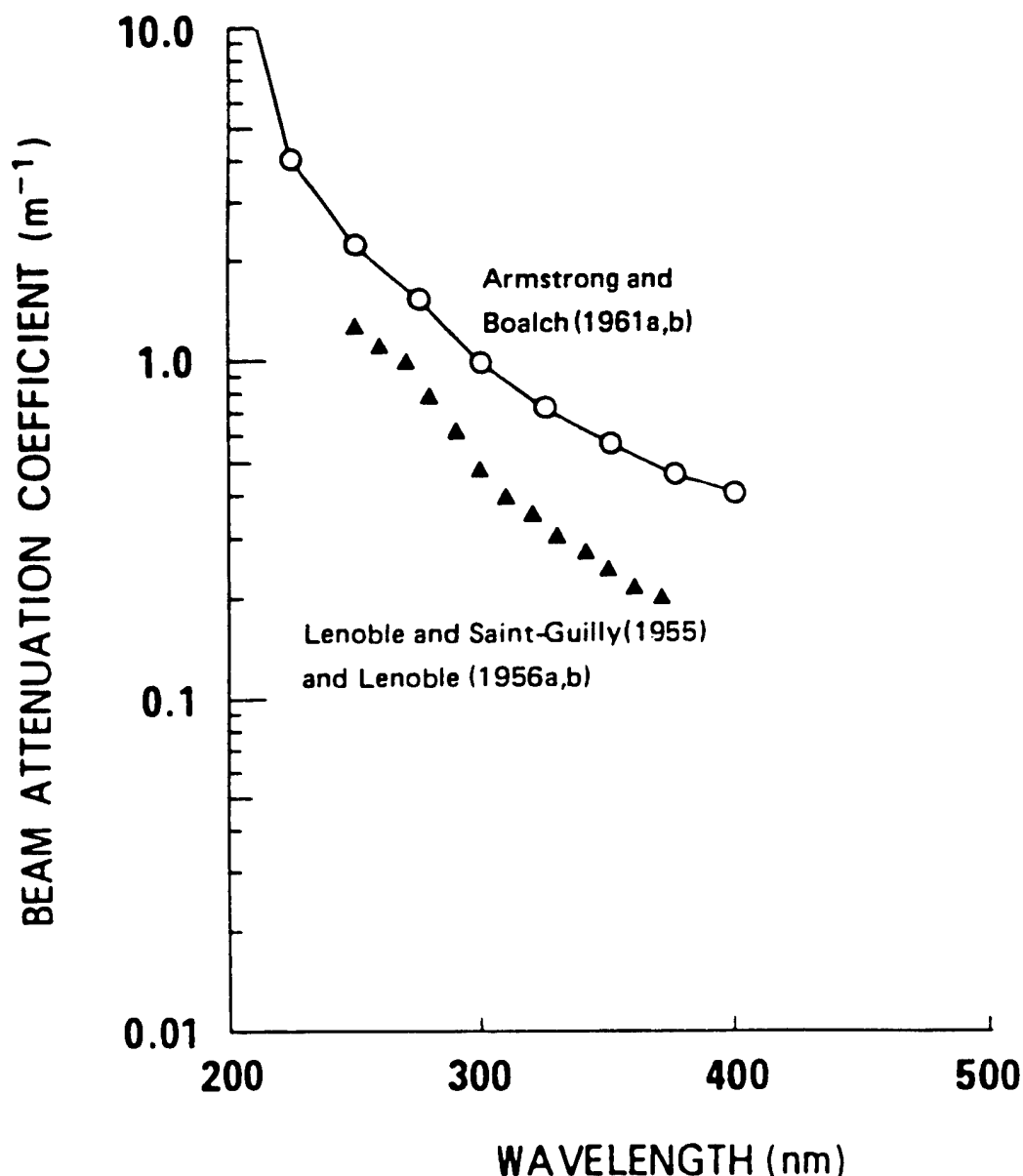


Figure 13. Typical absorption spectra of seawater. From *NRC* (1983).

Figure 14 shows some data for the effect of UV-B on several species of phytoplankton (*Worrest*, 1982). At first, the scatter appears to be quite bad, but further inspection of the figure shows that most of the scatter is due to variations between the different species. Note that the curve has no obvious curvature; i.e., there is no safe level of UV for these organisms. The point for 10% loss in carbon fixation for the average curve in Figure 14 is at a weighted fluence of 65 J/m^2 , which corresponds to about 2 h of normal sunlight at the equator at noon. It has been shown, however, that the action spectrum for phytoplankton damage is broader than that for cancer, so the RAF is less, and the sensitivity to ozone changes will be less (see, e.g., *Cullen and Neale*, 1994). A study of zooplankton mentioned in the same review (*Worrest*, 1982) shows a significant decline in the number of offspring of an irradiated generation of *Acartia Clausii*, a marine copepod.

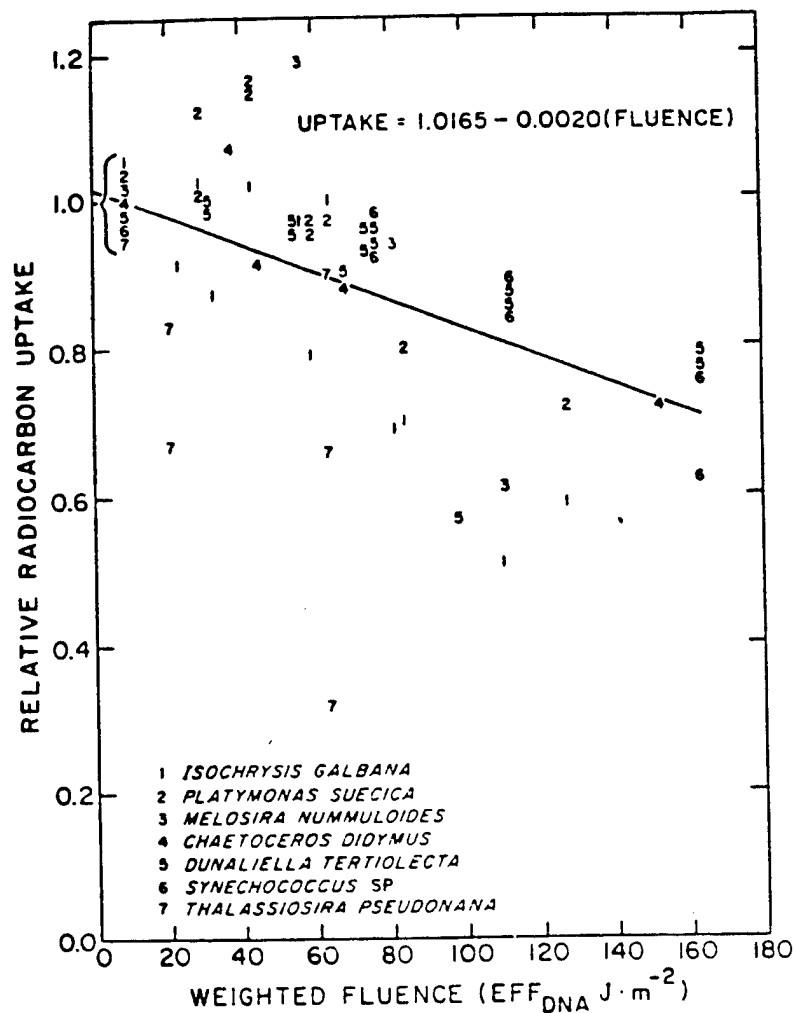


Figure 14. Effect of DUV on the photosynthesis rate (primary productivity) of seven species of phytoplankton. From Worrest (1982).

Other studies have shown that shrimp and crab larvae, and anchovies are sensitive to UV-B (ibid.)

Declines in primary productivity in the Antarctic due to the ozone hole have been documented (Smith, et al., 1992)

It appears that the RAF for phytoplankton photosynthesis inhibition is less than 1; probably about 0.3 (Cullen and Neale, 1994).

Haeder (1993) points out that in his estimate, a 16% loss of ozone would lead to a 5% loss in primary productivity (an implied RAF of 0.3), which in turn would lead to a 7% loss in fish production, or about 6 million tons of fish per year.

6. The Question of Adaptation

There remains in the community some controversy about whether or not organisms could compensate for, or adapt to, a higher level of UV radiation.

Studies have shown that both visible light-activated and dark repair mechanisms exist in plant and animal and human cells. Figure 15 shows the results of experiments on the UV-B survival rates for cultures of cells in which repair mechanisms have been genetically deleted (*Geise, 1976*). It can be seen that deletion of one repair mechanism (excision repair) increases mortality by a factor of 35; deletion of two repair mechanisms increases mortality by a factor of over 1000. It has been pointed out that without the known repair mechanisms, bacteria would be killed in only 10–20 s of ordinary sunlight (*NRC, 1976*). The significance of this is that repair mechanisms are already taking care of 99% to 99.9% of UV damage, and the harmful effects we see are due to unavoidable failures of the repair.

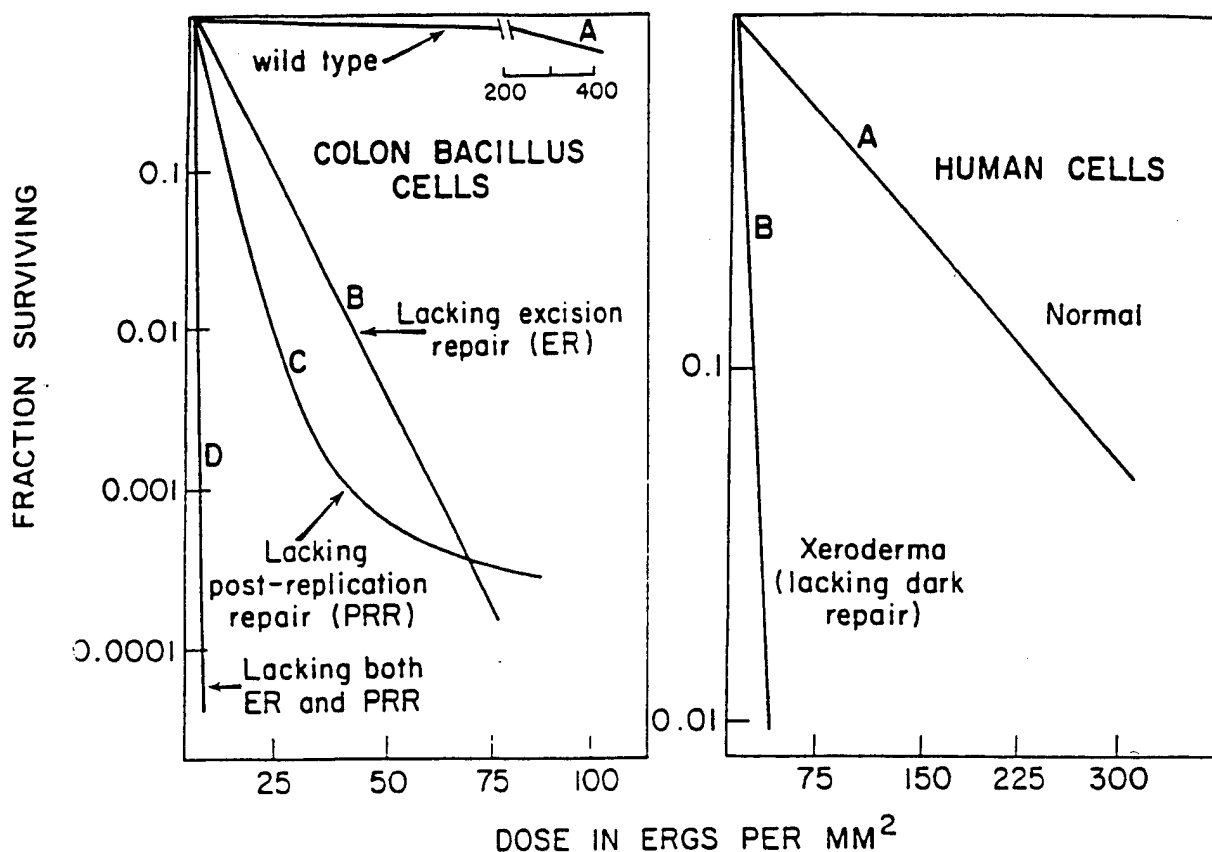


Figure 15. Plots comparing effects of repair mechanism deficient cells from bacteria (left) and humans (right). From *Geise (1976)*.

7. Conclusions

There are by now well-established connections between the introduction of chlorine-containing molecules into the stratosphere, a consequent decrease in the concentration of stratospheric ozone, and an increase in UV-B radiation at the surface of the Earth. An increase in UV-B on average would increase the incidence rate of non-melanoma skin cancer worldwide, with an unproved but likely increase in melanoma skin cancer. Other effects on humans could include cataracts and immune system inhibition, but these are less well established. The response of domestic and wild animal populations to UV-B is not thought to be a serious problem at this time, in part because they are protected by fur. The response of plants is complex because plants exist in a highly competitive situation with other plants for water and light, and must survive in an environment of pests and diseases which may also be affected by UV-B. The oceanic phytoplankton are the basis of the ocean food chain and are responsible for half of the natural carbon dioxide fixation. Studies have indicated that the phytoplankton are adversely affected by UV-B, and thus there is a potential for excess UV-B to affect fishing yields and the amount of carbon dioxide in the atmosphere.

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